Dear Mel.

Your letter of February 6 reached me the day before I left Cold Spring Tarbor for Austin, Texas, to give a set of lectures. I returned from Texas a week later with a blooming cold and much fatigue. Both conditions a e now relieved. They account, however, for the long delay in replying to your letter.

I was aslighted to receive an outline of your recent progress and very much pleased with the results. One aspect—the placement of the controlling element associated with we—resembles that of Judd with we and its derivatives. In both cases, the controlling element lies within a common region of the white locus. I noted this initially in reviewing Judd's abstract that appeared in the July, 1967, issue of Genetics. In a recent talk with Judd at Austin, I learned that in his case, he is finding so thing strange at the white locus in salivary chromosome examinations and he, also, is not able to interprete its exact nature. From this conversation I gathered that you know of his recent results and therefore, I need not recount them.

The similarity of placement of the "controlling element" in the $\underline{w}^{\textbf{c}}$ and $\underline{w}^{\textbf{zm}}$ cases conforms with the cases in whise where different known systems have taken over control of gene action at the wx locus. The tests of this were conducted by Oliver Melson. He is now writing up the results of a large number of tests aimed at placement of sites of change within the wx locus that are responsible for mutant expressions. Most of the mutants were "spontaneous" in origin. A few were produced by X-rays. Both multisite and single site mutants are represented among these selections. In addition, five cases of change in gene action induced by the presence of known controlling elements were included in the site mapping tests. One multisite (deficiency) mutant covers a segment within the locus The controlling element in each of the that is close to its middle. five cases lies within this region of the locus. Three of the five

cases are independent inceptions of control of action of the <u>Wx</u> gene by the <u>Ac</u> system. A fourth is a stable mutant derived from one of these. The fifth is an instance of control by the <u>Spm</u> system. When homozygous none of the five cases gives rise to a wild-type allele. Also, the stable mutant when combined with the mutable allele from which it arose gives no wild-type alleles. Otherwise, each combination gives rise to a few wild-type alleles. It has been possible to order the sites within the controlling element region that distinguish one allele from another. There is no evidence that the different alleles of the c.e. alter crossover frequencies within the locus when combined with alleles derived rom spontaneous mutation.

Melson plans to send the manuscript to me sometime this coming spring. I will then have more precise information. Just now, I have only the detailed information that was available from tests conducted up to April, 1966. These are the data that I showed to you this past June. Some of the more recent information given above comes from a telephone conversation that I had with Melson several weeks ago.

The west the needed desails, I will send you a susmary. I am sure Nelson will allow you to refer to his yet angublished work provided you send him a copy of your statement in order that no misrepresentation ampear in it. Feter Peterson has an abstract in the July, 1967, Genetics in which he refers to Nelson's results. His statements, however, were incorrect and misleading. This was disturbing to Nelson (and to me). Peterson did not check with Nelson before submitting the abstract.

Again, many thanks for keeping me informed. It is much appreciated. Also, give my very best to Katie.

As always.